Chapter 4
Echo Assessment of Systolic and Diastolic Function in Acute Coronary Syndrome

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Introduction

The human heart, being almost exclusively dependent on aerobic metabolism, requires a constant supply of oxygen to avoid tissue injury. Even at rest, the human myocardium extracts almost the entire oxygen content of the passing blood. This results in extremely low resting oxygen saturation in the coronary sinus, the final repository of the coronary blood (35% at rest; 25% at peak exercise). Therefore, the primary means of increasing oxygen delivery to the myocardium is through augmentation of coronary blood flow. From rest to maximal physical exertion, coronary flow increases up to fivefold.[1]

Although the pressure in the epicardial coronary arteries may vary significantly, the precapillary pressure in the myocardium is held almost constant at 45 mmHg thanks to autoregulation accomplished through dynamic changes in the arteriolar resistance.[2] Due to this autoregulation, a narrowing in an epicardial coronary artery has to be very severe (about 90% diameter loss) for the stenosis to become clinically evident at rest; blood supply limitation with exercise become evident when the stenosis reaches 70%.

Once the epicardial stenosis reaches a critical level, the loss of myocardial function and the development of clinical signs and symptoms proceed in an orderly fashion. This stepwise process is referred to as ischemic cascade.[3] It starts with an intramyocardial perfusion defect and progresses through a diminished left ventricular diastolic function, a decreased myocardial contractility, an increased left ventricular end-diastolic pressure, ST-segment changes, and ends, occasionally, with angina pectoris (Fig. 4.1).

Intramyocardial perfusion defects are the earliest sign of limitations in the coronary blood supply and can be detected by either myocardial contrast echocardiography (MCE) or nuclear imaging. MCE is discussed elsewhere in this textbook. In this

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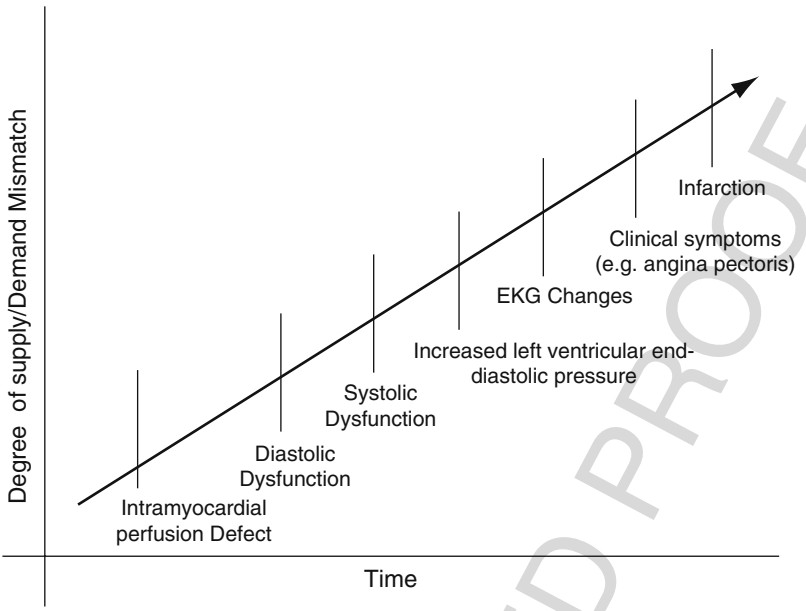

Fig. 4.1 Ischemic cascade. The loss of myocardial function and the development of clinical signs and symptoms proceed in a stepwise fashion as the cardiac demand increases

chapter, we will concentrate on the next two steps in the ischemic cascade, namely the loss of diastolic and systolic function during acute coronary syndromes.

Regional vs. Global Parameters of Dysfunction

Once the coronary supply/demand mismatch reaches a certain threshold level, there is a loss of normal myocardial function. The fundamental characteristic of ischemic dysfunction (either diastolic or systolic) is that it occurs regionally and that its distribution pattern conforms to the expected coronary blood supply of the 17-segment model discussed in Chapter 4. Conversely, when regional dysfunction is due to non-ischemic causes its distribution tends to be patchy and often spread over two or more coronary territories.

In the absence of extensive collaterals or surgical bypass grafting, the loss of myocardial function usually occurs first in the distal segments and spreads gradually toward the cardiac base. For instance, in a case of a proximal left anterior descending (LAD) artery stenosis, the first segments to lose function tend to be the apical ones followed by mid-cavity and basal segments.

When assessing myocardial systolic or diastolic dysfunction in acute coronary syndrome, one may evaluate regional abnormalities directly or measure their impact on the global ventricular function. Although diastolic dysfunction precedes the

systolic one in the ischemic cascade, we will discuss systolic dysfunction first since in routine clinical practice it is assessed in almost all patients. This is in contrast to diastolic dysfunction for which there is a much smaller body of echocardiographic evidence to guide the diagnosis, treatment, and prognosis.

Assessment of Regional Systolic Function in Acute Coronary Syndrome

Occlusion of an epicardial coronary artery at the time of acute coronary syndrome may lead to a loss of contractile function in the myocardial segments subtended by that artery. The magnitude and duration of such a contractile loss is dependent on both the severity and the duration of the coronary occlusion (Fig. 4.2).

In unstable angina, left and right ventricular wall motion is usually normal unless resting transthoracic echocardiography happens to be performed fortuitously during an episode of chest pain.

Non-ST elevation myocardial infarction (NSTEMI) usually results from an occlusion of a coronary branch vessel often in an elderly patient with preexisting collateral coronary circulation. Typically the loss of contractile function is restricted to the subendocardial layer which is most vulnerable to ischemia. However, on standard echocardiography the contractility loss will be observed in the entire thickness of the affected myocardial segment. This overestimation of contractile loss is attributed to tethering (an apparent passive loss of contractility in normal segments due to contractile loss in an adjacent area).

ST elevation myocardial infarction (STEMI) often results from an occlusion of a major coronary vessel and tends to occur in a younger age group compared to

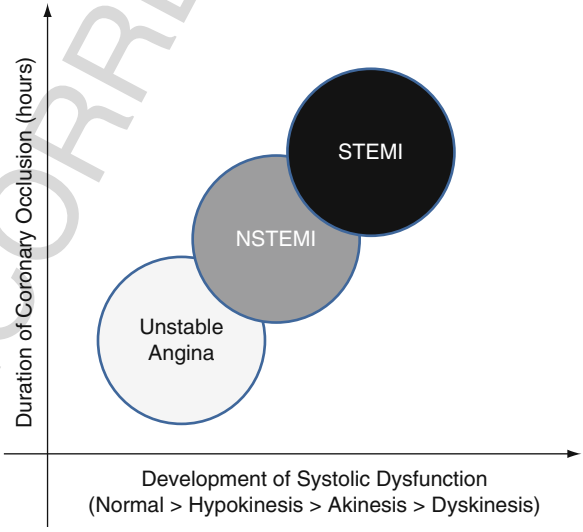

Fig. 4.2 Progression of myocardial dysfunction in acute coronary syndrome. Note that the magnitude and duration of myocardial systolic dysfunction is dependent on both the severity and the duration of coronary occlusion

NSTEMI. If the total session of coronary flow lasts for more than 6 h, myocardial necrosis will occur and the myocardium in the affected segments will be replaced with a fibrous scar over the ensuing weeks.

The magnitude of regional contractile loss in acute coronary syndrome is usually assessed semiquantitatively; one reports descriptively on the following three parameters:

1. Magnitude of contractile loss in each affected segment

NORMAL	Contractility preserved
HYPOKINESIS	Partial loss of contractility
AKINESIS	Complete loss of contractility
DYSKINESIS	Paradoxical movement of the affected segment away from The center of the ventricle during systole
ANEURYSMAL	Outward movement of the affected segment during both Systole and diastole

2. Number and location of affected segments
3. Suspected coronary artery distribution (left anterior descending artery vs. right coronary artery vs. left circumflex artery)

Wall scoring provides a more rigorous quantitative approach to assessing wall motion abnormalities in acute coronary syndrome. However, the wall scoring method assesses the contractility of all ventricular segments and is thus described in the next section.

Assessment of Global Systolic Function in Acute Coronary Syndrome

Global ventricular systolic function in acute coronary syndrome may be assessed through either wall motion scoring or calculation of global ventricular ejection fraction.

Wall Motion Scoring

Wall motion scoring analysis assigns a numeric value to the degree of contractile dysfunction in each segment. The actual numeric values given to particular forms of contractile (dys)function vary in the published literature; the most common scheme is given in Table 4.1.

Once all segments are given individual scores, a total score is calculated as a sum of individual scores. A wall motion score index (WMSI) in then calculated as a ratio between the total score and the number of evaluated segments. The WMSI is a dimensionless number; its range of values depends on the scoring scheme used. For the scoring scheme shown in Table 4.1, the WMSI would range between 1 and 5.

Table 4.1 Left ventricular wall motion scoring

	Score
Normal	1
Hypokinesis	2
Akinesis	3
Dyskinesis	4
Aneurysmal	5

$$\text{Wall motion score index} = \frac{\text{Sum of individual segment scores}}{\text{Number of evaluated segments}}$$

For a fully visualized normal ventricle, the total score is 17 (all segments have normal contractility). Since all 17 segments are evaluated, the wall score index of a normal heart is 17/17 = 1. For abnormal ventricles, the higher the WMSI, the more the contractile dysfunction. The theoretical maximum for a WMSI is 5 in the scoring scheme depicted in Table 4.1; such a score would assume that all left ventricular segments are aneurysmal, a condition incompatible with life. Between the extremes of 1 and 5 are the values obtained in patients with acute coronary syndrome.

Using the same methodology, one can use the 16-segment model instead of the 17-segment one. The underlying notions will not change: the higher the WMSI, the worse the systolic dysfunction. For example, in a patient with acute coronary syndrome who had a total occlusion of the proximal LAD, akinesis was observed in the entire apical region (segments 13, 14, 15, and 16), while hypokinesis was observed in the remaining LAD territory (segments 1, 2, 7, and 8). Segments in the territories of other coronaries were normal. This patient's global WMSI was calculated as [4(3) + 4(2) + 8(1)]/16 = 1.75 (Fig. 4.3).

Instead of a global WMSI, one can also calculate a regional WMSI taking into account only segments supplied by a particular artery. For the patient above, the regional LAD score would be [4(3) + 4(2)]/8 = 2.5 (Fig. 4.3). Because of tremendous variability in the size of RCA and LCx territories between patients, it is often more prudent to provide a regional score for the entire non-LAD (RCA + LCx) territory rather than individual scores for RCA and LCx when there is no prior knowledge of a coronary dominance pattern in an individual patient.

A major shortcoming of the above WMSI analysis is that it does not include right ventricular wall segments despite the fact that the presence of right ventricular systolic dysfunction may portend a worse prognosis in patients with acute coronary syndrome.[4]

Assessment of Ventricular Ejection Fraction

Numerous studies have shown that the left ventricular ejection fraction (LVEF) is one of the most powerful predictors of future mortality and morbidity in patients

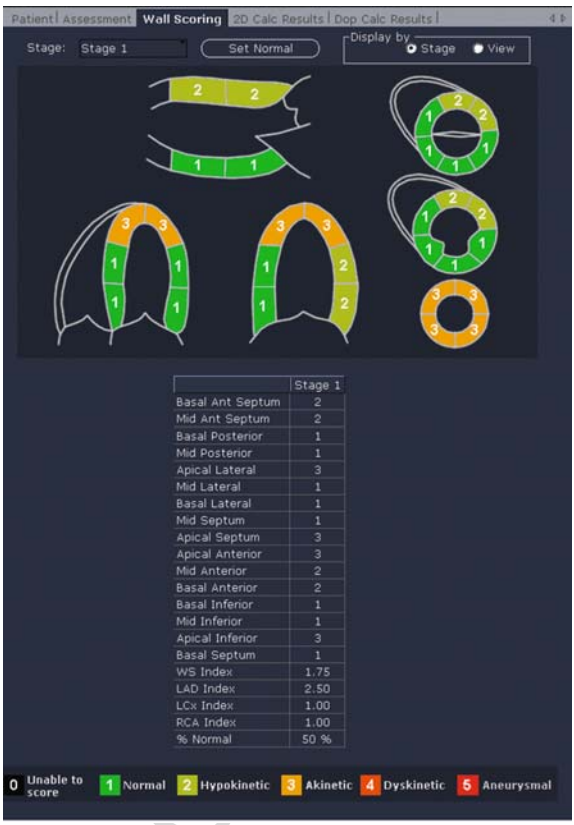

Fig. 4.3 Wall motion score index (WMSI) calculations using a 16-segment left ventricular model. This patient with acute coronary syndrome had a total occlusion of his proximal left anterior descending (LAD) artery leading to akinesis of the four apical segments and hypokinesis in the basal and mid segments of the anterior wall and the anterior septum. Other left ventricular segments were normal. Note the global WMSI (WS Index) of 1.75, and the regional LAD score (LAD Index) of 2.50. Note also that the regional scores were normal (1.00) for both the right coronary artery (RCA) and the left circumflex (LCx) artery; this indicates that the wall motion abnormalities in this patient were confined to the LAD territory

with left ventricular systolic dysfunction of any cause including ischemic heart disease.[5] For instance, LVEF is the single most powerful predictor of mortality and the risk for life-threatening ventricular arrhythmias after myocardial infarction.[6] Furthermore, once the acute coronary syndrome resolves, the residual LVEF is important for treatment as LVEF cutoff values are built into recommendations for both medical and electrical device therapies. Even with treatment and clinical stabilization of heart failure, there is an inverse, almost linear, relationship between LVEF and survival in patient whose LVEF is less than 45% (Fig. 4.4).[7]

By definition, LVEF is the percentage of the end-diastolic volume that is ejected with each systole as the stroke volume. Thus, to calculate the LVEF one needs to estimate the end-systolic and end-diastolic volume of the left ventricle.

Current recommendations of the American Society for Echocardiography and the European Association for Echocardiography discourage the use of M mode-derived methods such as the cube rule for calculation of left ventricular volumes.[8] M mode is particularly ill-suited for estimating LVEF in patients with ischemic heart disease involving the apical regions of the left ventricle because M mode measurements are made at the base of the heart; the calculated regional LVEF at the mid-papillary level

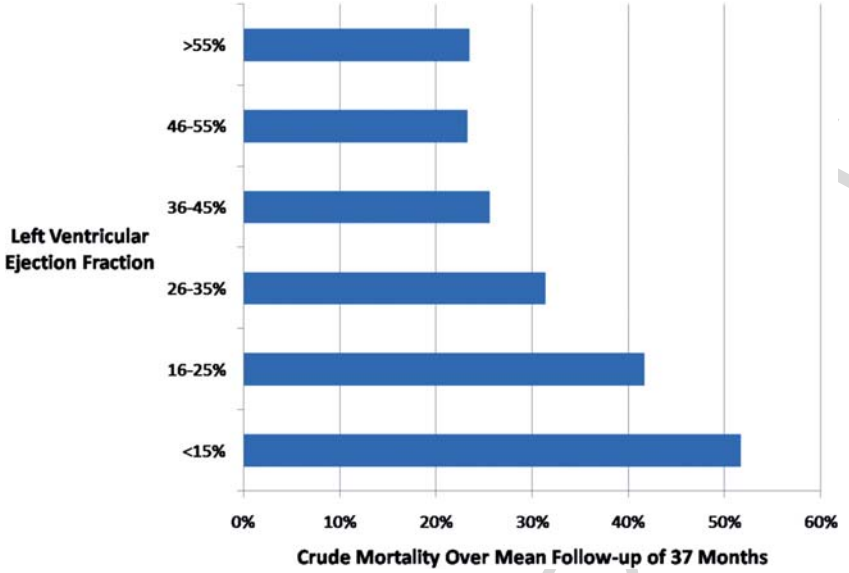

Fig. 4.4 Relationship between left ventricular ejection fraction and survival. Note the negative almost linear relationship between survival and left ejection fractions <45%. Based on numeric data from Curtis et al.[7]

is clearly not representative of the global LVEF in patients with apical wall motion abnormalities.

For two-dimensional echocardiography, biplane Simpson's rule is the gold standard for estimation of the LVEF[9] Most modern ultrasound systems provide a semi-automated software package for the Simpson's rule analysis. Operators are usually required only to trace the left ventricular border of an end-diastolic and an end-systolic frame in the apical four-chamber and two-chamber views; the software package then automatically calculates the left ventricular end-diastolic volume, end-systolic volume, and LVEF (Fig. 4.5). One should be aware, however, that when mitral or aortic regurgitation is present, Simpson's rule calculates the total stroke volume which is the sum of the regurgitant volume and the true antegrade stroke volume; therefore, the calculated LVEF, although technically correct, may not be a good measure of left ventricular systolic performance.

With the advent of real-time three-dimensional (RT3D) transthoracic techniques, left ventricular volumes and LVEF can now be calculated with even greater accuracy than is possible with the biplane Simpson's rule (Fig. 4.6). RT3D-derived left ventricular volume data are now comparable to those obtained by cardiac magnetic resonance imaging, the prior gold standard for such calculations.[10]

In conclusion, whenever available, left ventricular volumes and LVEF in acute coronary syndrome should be calculated from an RT3D system; the biplane Simpson's rule should be the next best method for such calculations when only a two-dimensional ultrasound system is available.

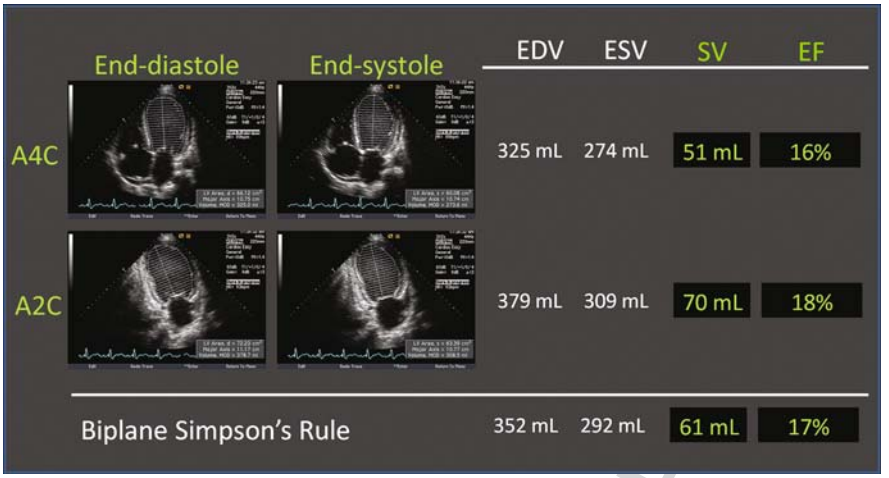

Fig. 4.5 Calculation of left ventricular ejection fraction (LVEF) by biplane Simpson's rule. The operator of an ultrasound system is required to trace the endocardial border of an end-diastolic and an end-systolic frame in the apical four-chamber (A4C) and two-chamber (A2C) views. The system then calculates the end-diastolic volume (EDV), end-systolic volume (ESV), stroke volume (SV), and LVEF

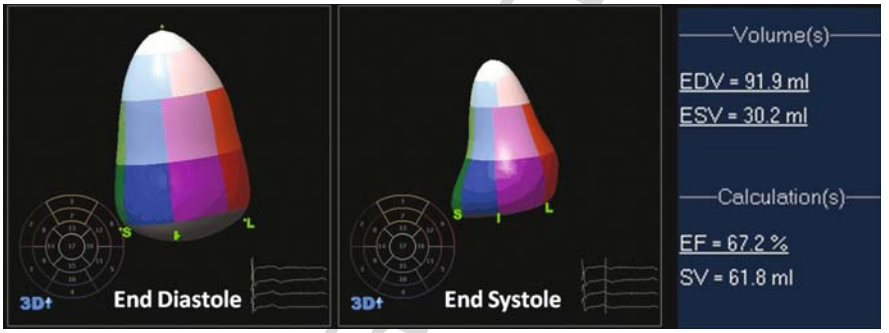

Fig. 4.6 Calculation of left ventricular volumes and ejection fraction (EF) by three-dimensional echocardiography. A 3D ultrasound system calculates the end-diastolic volume (EDV), end-systolic volume (ESV), stroke volume (SV), and LVEF automatically from a 3D data set after an operator manually enters key reference points of the left ventricle

Comparing Wall Motion Scoring to Left Ventricular Ejection Fraction

A major feature of the wall scoring system is that it does not differentiate between normal and hyperdynamic left ventricular segments. This may be viewed as either an advantage or a shortcoming of the wall scoring method.

Let us take an example of two patients with acute coronary syndrome both of which have hypokinesis in all LAD segments. In one patient, however, the left

ventricular segments in the non-LAD territory are hyperdynamic, while in the other patient they move normally. According to the wall scoring method described above, both would have the same wall motion score index yet their LVEF would be different (LVEF is expected to be higher in the first patient). WMSI in this case accurately reflects the extent of wall motion abnormalities due to acute coronary syndrome in the two patients but is unable to take into account the compensatory hyperkinesis in the second patient the way global LVEF can.

Strain Imaging in Acute Coronary Syndrome

Wall motion scoring described above relies on subjective 'eyeballing' of left ventricular thickening and wall motion during the cardiac cycle and thus requires a large degree of experience and expertise. Strain imaging has recently entered the armamentarium of echocardiography and promises to provide a more objective and quantitative basis for wall motion analysis.

Strain imaging is based on the fact that each of the 17 segments in the left ventricular model changes its length throughout the cardiac cycle. In the longitudinal direction, each segment *shortens* from end diastole to peak systole; this can be observed in apical four-chamber, two-chamber, and three-chamber views. In the radial direction, each segment *shortens* (*thickens*) from end diastole to peak systole; this can be observed in any of the short-axis views of the left ventricle. From peak systole to end diastole, the process reverses: each ventricular segment *lengthens* in the longitudinal direction and *shortens* (*thins*) in the radial direction.

Strain is a unitless ratio between the segment length at any point in the cardiac cycle and the reference length at end diastole. In other words, strain is a fractional change in the segment length during the cardiac cycle. Because left ventricular segments *lengthen*in the longitudinal direction, their longitudinal systolic strain has a negative value. This is in contrast to radial strain which has a positive value in systole due to wall thickening. The opposite is true for both longitudinal and radial strain during diastole. Echocardiographically, strain data are obtained from either tissue Doppler velocity data or speckle tracking.[11]

In a normally contracting left ventricular segment, peak strain value is achieved just prior to aortic valve closure. In patients with unstable angina or non-ST elevation (nontransmural) infarction, two changes occur: the magnitude of systolic strain diminishes and the peak strain occurs progressively later well past the aortic valve closure. The latter phenomenon is referred to as 'postsystolic thickening' and is still poorly understood despite decades of experimental work in animal models. It is important to emphasize that postsystolic thickening is a sensitive but not a specific sign of ischemia; it may also be observed in other disorders such as myocardial storage diseases and in states of high left ventricular afterload (such as aortic stenosis and elevated systemic blood pressure).[11]

In ST elevation (transmural) infarction with nonviable myocardium, no active strain is present and may be replaced with outward bulging (dyskinesis). Strain pattern in normal and ischemic myocardium is summarized diagrammatically in Fig. 4.7.

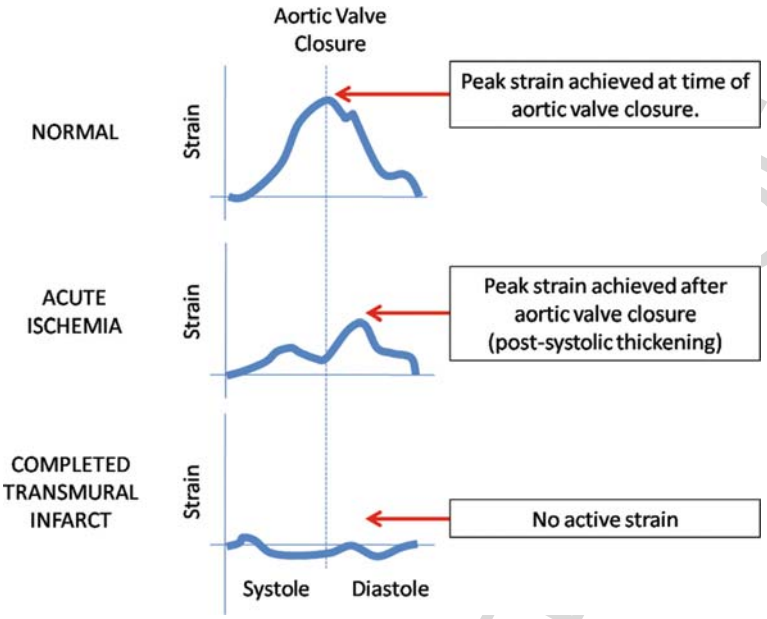

Fig. 4.7 Patterns of left ventricular strain in normal and ischemic myocardium. Schematic representation of radial strain recordings. Note that in the normal myocardium, peak systolic strain occurs at the time of aortic valve closure. During ischemia, the magnitude of systolic strain diminishes and the peak strain occurs past the aortic valve closure (postsystolic thickening). In the fully infracted myocardium, there is no active systolic or postsystolic strain. Drawn based on data from Bijnens et al.[11]

Assessment of Diastolic Function in Acute Coronary Syndrome

In patients with acute syndrome, assessment of left ventricular diastolic function should follow the general guidelines of echocardiographic analysis of diastolic parameters. The analysis should include at least the following three aspects:

1. Evaluation of the pattern of mitral and pulmonary venous blood flow velocity determined by pulsed wave Doppler.
2. Measurement of diastolic mitral annular tissue excursion using tissue Doppler techniques.
3. Calculation of the left atrial volume.

It is important to emphasize that the diastolic changes described in this chapter are not specific to acute coronary syndrome and occur in a wide variety of cardiac and extra-cardiac disorders (renal failure, anemia, high afterload due to stiff aortic tree, etc.).

Mitral and Pulmonary Venous Blood Flow Velocity Pattern

In young individuals, left ventricular filling occurs primarily during the early (E) phase of diastole with only a minor contribution from atrial contraction in late diastole (A phase). Furthermore, the filling of the left atrium from the pulmonary veins is more prominent during ventricular diastole (D wave) and during ventricular systole (S wave) and the atrial reversal of flow (AR wave) from the left atrium into the valveless pulmonary veins during atrial contractions is small. In summary, in a young individual the diastolic pattern is characterized by mitral E wave dominance, pulmonary vein D wave dominance, and a small AR (Fig. 4.8).

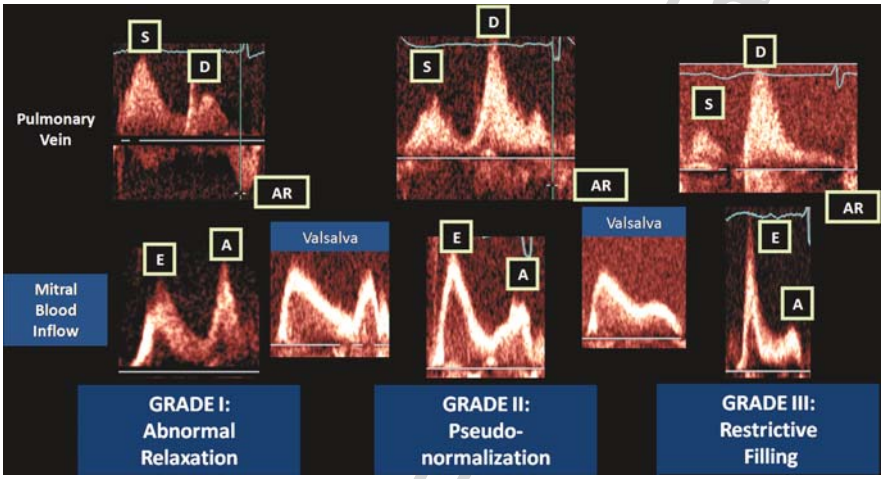

Fig. 4.8 Mitral and pulmonary venous filling patterns. *Top panel* shows pulmonary vein tracings; S, systolic wave; D, diastolic wave; AR, atrial reversal wave. *Bottom panel* shows mitral blood inflow tracings; E, early diastolic wave; A, atrial kick

The amplitude (peak velocity) of the mitral E wave is governed by the pressure gradient between the left atrium and the left ventricle in early diastole; similarly the magnitude of the pulmonary D wave is determined by the pressure gradient between the pulmonary veins and the left ventricle during the early period of ventricular diastole.

In young individuals, these gradients are characterized by very low ventricular pressures and flow from the pulmonary veins and the left atrium driven by ventricular suction.

In humans, the 'normal' aging process is characterized by a loss of relaxing properties in the left ventricle during diastole. Due to slowed relaxation, the left ventricular pressure remains relatively high during early diastole which in turn diminishes the left atrial-to-left ventricular and pulmonary venous-to-left ventricular gradients during early diastole. As a consequence, the amplitude of the E wave and the pul-

monary venous D wave diminishes progressively, while the deceleration of the E wave prolongs. In an elderly person, the pattern of diastolic flow thus becomes A dominant, S dominant, and with a prominent AR wave (both in amplitude and duration). This pattern has been termed abnormal relaxation or grade I (mild) left ventricular dysfunction.

Left ventricular relaxation during early diastole is an active, energy-consuming process requiring a continuous supply of oxygen. It can therefore be expected that in acute coronary syndrome left ventricular relaxation is impaired; indeed such impairment precedes systolic dysfunction in the ischemic cascade (Fig. 4.1). Using the pulsed wave Doppler mitral inflow velocity pattern, one can easily show transition from an E dominant pattern at baseline to an A dominant pattern with prolonged E wave deceleration time within seconds of acute coronary occlusion.[12] In humans, this is infrequently observed because most acute coronary syndromes occur in late middle-age and elderly patients who have the pattern of abnormal relaxation at baseline due to 'normal' aging.

When relaxing properties are severely impaired in moderate and severe left ventricular dysfunction, there is a compensatory increase in the left atrial pressure (preload) in an attempt to normalize the filling pressure gradient. As a result, the magnitude of the E and D waves rises in proportion to the rise in the left atrial pressure. In moderate diastolic dysfunction, the combination of abnormal left ventricular relaxation and moderately elevated left atrial pressure gives rise to the so-called pseudonormal filling pattern (E dominant, D dominant with an E wave deceleration time >150 ms; grade II diastolic dysfunction). Severe diastolic dysfunction is characterized by ever taller E and D waves but with an E wave deceleration time <150 ms and is referred to as restrictive filling (grade III diastolic dysfunction).

Pseudonormal and restrictive filling patterns are combinations of diminished left ventricular relaxing properties (left ventricular dysfunction) and elevated preload (elevated left atrial pressures). The Valsalva maneuver diminishes preload and unmasks the underlying left ventricular relaxation abnormalities. After the Valsalva maneuver the pseudonormal pattern will become the abnormal relaxation pattern; this is important in distinguishing a normal from a pseudonormal pattern.

Pseudonormal and restrictive filling patterns are frequently encountered in patients with acute coronary syndrome, especially when there is concomitant systolic dysfunction and diminished left ventricular ejection fraction. When such patterns are observed, they are indicative of elevated left atrial pressures and should alert a clinician to actively pursue the diagnosis and treatment of pulmonary edema. This is further discussed in the section on mitral annular tissue Doppler.

After a Valsalva maneuver, restrictive filling pattern will often revert to a pseudonormal pattern (reversible restrictive filling pattern). When this fails to occur (irreversible restrictive filling pattern), the prognosis is very poor.[13]

In summary, grade I left ventricular dysfunction is indicative primarily of left ventricular dysfunction, while grades II and III (pseudonormal and restrictive filling patterns) are primarily indicative of elevated left atrial pressures.

Mitral Annular Tissue Doppler Analysis

After placing a pulsed Doppler sample volume at the level of either septal or lateral mitral annulus in the apical four-chamber transthoracic view, one obtains E and A waves similar to the mitral blood velocity pattern described above except that the mitral annular tissue Doppler waves move in the direction opposite to the blood flow. These annular waves are often labeled E′ and A′ to distinguish them from the equivalent mitral blood velocity waves (Fig. 4.9).

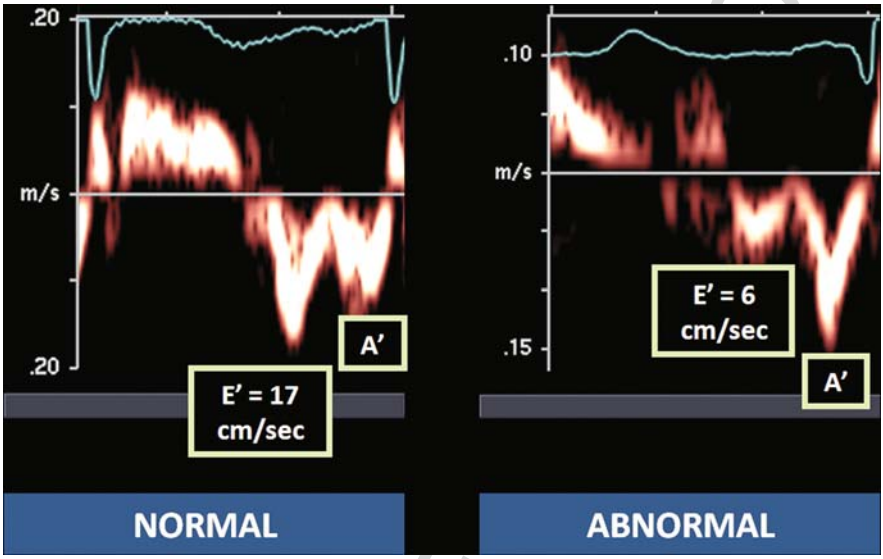

Fig. 4.9 Normal and abnormal mitral annular tissue Doppler tracings. *Left panel* shows a normal pattern; *right panel* reveals diminished E′ velocity consistent with abnormal left ventricular relaxation

The amplitude (peak velocity) of E′ is inversely related to left ventricular relaxing properties (the lower the E′ velocity, the greater the left ventricular dysfunction). In the elderly, peak E′ velocities of less than 8 cm/s is abnormal; in young individuals the cutoff value of 10 cm/s is used. Mitral tissue Doppler E′ measures primarily the diastolic properties of the left ventricle and is relatively preload independent (unaffected by left atrial filling pressures); this is in contrast with mitral and pulmonary venous blood velocities, which simultaneously reflect both the left ventricular diastolic properties and the left atrial filling pressures.

Clinically, the most useful application of the mitral annular tissue Doppler analysis is the ratio of the mitral blood velocity E wave and the mitral annular E′ velocity. An E/E′ ratio of <8 is indicative of normal filling pressures. An E/E′ ratio >15 implies elevated filling pressures. When one observes an E/E′ >15 in a patient with acute coronary syndrome, there is a strong possibility that the patient is in pulmonary edema. When E/E′ values range between 8 and 15, left atrial pressure

may be either normal or elevated.[14] In addition, estimation of left ventricular filling pressures by E/E′ ratio is a powerful predictor of survival after acute myocardial infarction; the higher the E/E′ ratio, the lower the survival.[15]

Left Atrial Volume

Usually, left atrial volume does not change precipitously in patients with acute coronary syndrome. However, chronic remodeling over weeks and months after completed myocardial infarction leads to progressive left atrial enlargement due to chronically elevated left atrial pressures. Conversely, in the absence of significant mitral and aortic valve disease, the mere finding of increased left atrial volume is indicative of abnormal left ventricular filling characterized by chronically elevated left atrial pressures.

Left atrial volumes are usually calculated using the area-length method and indexed for body surface area (Table 4.2). The same reference values are used for both women and men.

Table 4.2 Normal and abnormal left atrial volumes

Left atrial size	Left atrial volume indexed to body surface area (mL/m^2)
Normal	22 ± 6
Mildly enlarged	29–33
Moderately enlarged	34–39
Severely enlarged	≥ 40

Based on data from Lang et al.[8]

Conclusion

In patients with acute coronary syndrome, assessment of left ventricular function should be based on the 17-segment model for both standard wall motion analysis and strain imaging. Assessment of left ventricular diastolic function should include mitral inflow and pulmonary vein blood velocity pulsed Doppler recordings, mitral annular tissue Doppler tracings, E/E′ ratio, and calculation of the left atrial volume indexed to patient's body surface area.

Clinical Cases

Gerard Oghlakian, MD, Ramzan Zakir, MD, and Christine Gerula, MD of New Jersey Medical School in Newark, NJ have contributed to the following cases.

Clinical Case #1

Subjective

A 72-year-old man with history of hypertension, diabetes mellitus, and coronary artery disease (CAD) presented to the emergency room complaining of intermittent chest pain of 5 days duration. He described the chest pain as being left-sided, nonexertional, waxing and weaning, and lasting few minutes at a time. He denied concomitant shortness of breath, nausea, or vomiting. There was no diaphoresis, lightheadedness, or syncope.

His past medical history of CAD consisted of a previous myocardial infarction and stent placement in the distal left anterior descending (LAD) artery 1 year ago. He also had history of mechanical aortic valve replacement 10 years prior. He denied any tobacco, alcohol, or drug abuse. He was compliant with all his medications including an angiotensin-converting inhibitor, a beta blocker, a statin, and aspirin.

Objective

In the emergency room, his physical exam revealed a blood pressure of 110/73 mmHg and a heart rate of 89 beats per minute. His respiratory rate was 22 respirations per minute and his oxygen saturation was 99% on room air. He did not appear to be in any distress. He had a normal jugular venous pressure. His lungs were clear to auscultation. His cardiac auscultatory findings were normal. There was no peripheral edema.

The electrocardiogram obtained in the emergency department showed normal sinus rhythm, inferior Q waves, and lateral T wave abnormalities consisting of a slight ST depression in the precordial lead V_6 and T wave inversions in leads I and aV_L (Fig. 4.10).

Assessment and Plan

A presumptive diagnosis of cardiac biomarker-negative unstable angina was established.

Indication for the Echo

While still in the emergency department, the patient had another episode of chest pain. His electrocardiogram obtained during the chest episode remained unchanged from the baseline. A stat echocardiogram was performed in the emergency department while the patient was still having chest pain.

Echo Imaging

Transthoracic echocardiography at the time of chest pain revealed hypokinesis of the basal and mid segments of the inferior and posterior (inferolateral) walls (segments

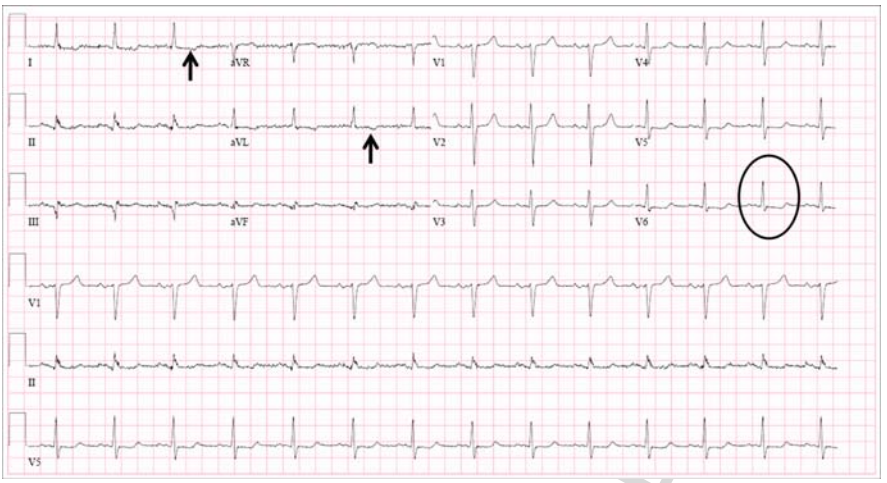

Fig. 4.10 Electrocardiogram (EKG). EKG reveals normal sinus rhythm, inferior Q waves, and lateral T wave abnormalities consisting of a slight ST depression in the precordial lead V_6 (*circles*) and T wave inversions in leads I and aV_L (*arrows*)

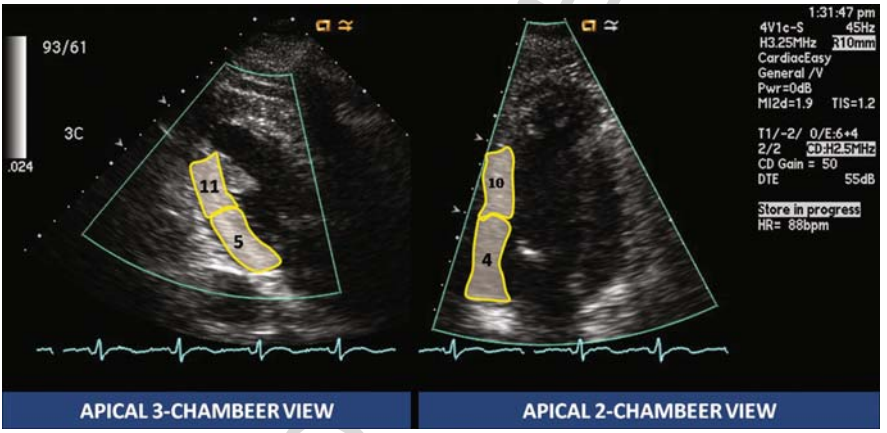

Fig. 4.11 Transthoracic echocardiogram. Transthoracic echocardiography at the time of chest pain revealed hypokinesis of the basal and mid segments of the inferior and posterior (inferolateral) walls (segments 4, 5, 10, and 11). There were no other left ventricular wall motion abnormalities including the regions supplied by the distal LAD (segments 13–17) where in-stent restenosis had occurred

4, 5, 10, and 11). There were no other left ventricular wall motion abnormalities including the distal anterior wall and the apex (segments 13–17). Left ventricular ejection fraction was diminished and was calculated at 40% (Fig. 4.11).

Management

Patient was taken to the cardiac catheterization laboratory. His coronary angiogram revealed that the right coronary artery was dominant and that it had no significant stenosis. In the distal left anterior descending (LAD) artery, there was in-stent restenosis. Just proximal to the takeoff of the first obtuse marginal branch, there was a 95% diameter stenosis of the left circumflex (LCx) artery (Fig. 4.12).

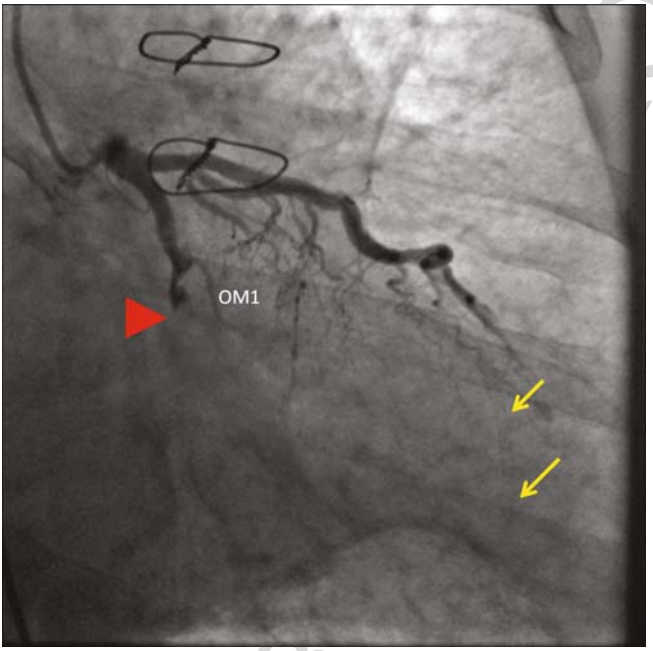

Fig. 4.12 Coronary angiogram. Note the abnormalities in both the LAD and the LCx. In the distal LAD, there is in-stent restenosis (*arrows*). Just proximal to the takeoff of the first obtuse marginal branch (OM_1), there is a 95% diameter stenosis of the LCx (*arrow head*). The image is obtained in the right anterior oblique (RAO) view (–27°) with caudal angulation (–30°)

Given the echocardiographic findings, the LCx was deemed to be the culprit vessel and the stenosis was successfully treated with the deployment of a drug-eluting stent. Despite restenosis of the stent in the distal LAD, there were no wall motion abnormalities in the LAD distribution likely due to natural collaterals.

Outcome

Following the percutaneous intervention, his chest pain resolved and he was discharged home. He remained chest pain free on subsequent follow-up.

Clinical Case #2

Subjective

A 69-year-old man with history of hypertension was found unresponsive in a local park. Emergency medical service was called and patient was transported to the hospital. No details of his past medical history could be obtained as the patient was unresponsive and no relative or friend could be contacted.

Objective

In the emergency department, his blood pressure was 185/65 mmHg and a heart rate of 55 beats per minute. His respiratory rate was 10 respirations per minute and his oxygen saturation was 99% on a 100% nonrebreather mask. There was no evidence of trauma. He had a normal jugular venous pressure. Patient was intubated for airway protection and auscultatory exam of the lungs was difficult. His cardiac exam revealed no murmurs, rubs, or gallop. He did not have any peripheral edema.

An electrocardiogram performed in the emergency department revealed sinus bradycardia, left atrial enlargement, and lateral T wave inversions (Fig. 4.13). Basic laboratory exam was remarkably for elevated serum glucose level (239 mg/dL; normal <109 mg/dL).

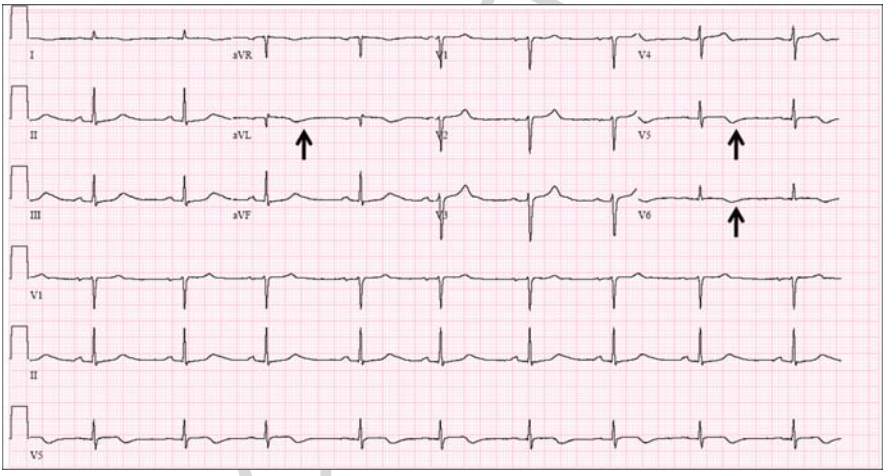

Fig. 4.13 Electrocardiogram (EKG). EKG reveals sinus bradycardia, left atrial enlargement, and lateral T wave inversions (*arrows*)

Serum troponin I peaked at 20.8 ng/mL (normal <0.4 ng/mL). Brain natriuretic peptide (BNP) was elevated at 1550 pg/mL (normal <100 pg/mL). Chest radiograph revealed pulmonary vascular congestion (Fig. 4.14).

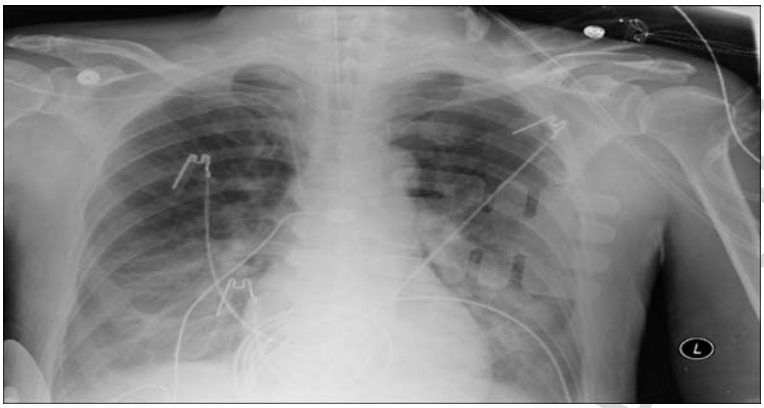

Fig. 4.14 Chest radiograph. Pulmonary vascular congestion is present in both lungs

Indication for the Echo

A presumptive diagnosis of acute coronary syndrome complicated by an acute pulmonary edema was established and transthoracic echocardiogram was ordered to assess left ventricular systolic and diastolic function.

Echo Imaging

Transthoracic echocardiogram revealed severe global LV systolic dysfunction. Assessment of left ventricular diastolic dysfunction revealed a restrictive filling pattern (grade III left ventricular diastolic dysfunction) based on mitral and pulmonary venous flow velocity recordings. Peak velocity of the mitral annular tissue Doppler E′ wave was low (6 cm/s) indicative of diminished left ventricular relaxation. E/E′ ratio was greater than 15 indicative of elevated left atrial pressures (Fig. 4.15). In

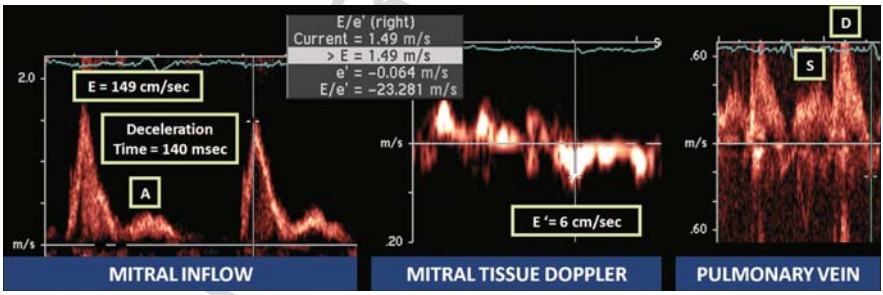

Fig. 4.15 Echocardiogram. Mitral inflow blood velocity pattern reveals restrictive filling pattern. Pulmonary venous flow with S < D is consistent with such a pattern. Peak velocity of the mitral annular tissue Doppler E′ is low. E/E′ ratio is greater than 15 indicative of elevated left atrial pressures and consistent with the clinical diagnosis of congestive heart failure

summary, echocardiographic findings were consistent with the clinical diagnosis of congestive heart failure (pulmonary vascular congestion on chest radiograph; highly elevated BNP).

Management

Patient was treated with an angiotensin-converting enzyme, a beta blocker, a statin, and an intravenous diuretic. His oxygenation improved and pulmonary vascular congestion resolved.

Outcome

The patient's neurologic status did not improve significantly and he continued to be in a persistent vegetative state and ventilator dependent. He had a tracheostomy and gastrostomy tube placed and was transferred to a long-term facility. He had no further cardiac evaluation or intervention in view of his poor neurologic outcome.

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Chapter 4

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